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=> file medline biosis caplus embase
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                                 0.22       0.22

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=> (growth(w)hormone or GH) and (intramusc? or subcutaneous?) and
(blood(w)brain(w)barrier or BBB)
(GROWTH(W)HORMONE IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
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"HELP COMMANDS" at an arrow prompt (=>).

=> s (growth(w)hormone or GH) and (intramusc? or subcutaneous?) and
(blood(w)brain(w)barrier or BBB)
L1          29 (GROWTH(W) HORMONE OR GH) AND (INTRAMUSC? OR SUBCUTANEOUS?) AND
               (BLOOD(W) BRAIN(W) BARRIER OR BBB)

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2           21 DUP REM L1 (8 DUPLICATES REMOVED)

=> dis ibib abs l2 1-21

L2   ANSWER 1 OF 21  EMBASE  COPYRIGHT (c) 2010 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:   2009519322  EMBASE
TITLE:              Cognitive enhancing effects of ghrelin receptor agonists.
AUTHOR:             Atcha, Zeenat (correspondence); Chen, Woei-Shin; Ong, Agnes
                    B.; Wong, Fong-Kuan; Neo, Aveline; Browne, Edward R.;
                    Pemberton, Darrel J.
CORPORATE SOURCE:   GlaxoSmithKline RandD China, Centre for Cognition and
                    Neurodegeneration Research, Helios Building, #03-01/02,
                    Biopolis at One-North, 11 Biopolis Way, Singapore 138667,
                    Singapore. Zeenat.I.Atcha@gsk.com
AUTHOR:             Witherington, Jason
CORPORATE SOURCE:   Department of Medicinal Chemistry, Immunoinflammation CEDD,
                    GlaxoSmithKline Medicines Research Centre, Gunnels Wood
                    Road, Stevenage, Hertfordshire SG1 2NY, United Kingdom.
AUTHOR:             Pemberton, Darrel J.
CORPORATE SOURCE:   Neurosciences Division, Johnson and Johnson PRD,
                    Turnhoutseweg 30, Beerse 2340, Belgium.
SOURCE:             Psychopharmacology, (2009) Vol. 206, No. 3, pp. 415-427.
                    Refs: 47
                    ISSN: 0033-3158; E-ISSN: 1432-2072  CODEN: PSCHDL
PUBLISHER:          Springer Verlag, Tiergartenstrasse 17, Heidelberg, D-69121,
                    Germany.
COUNTRY:            Germany
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DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 008 Neurology and Neurosurgery
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 11 Nov 2009
Last Updated on STN: 11 Nov 2009

AB Rationale: Ghrelin, the endogenous ligand for the growth hormone secretagogue receptor, has been shown to play a role in multiple physiological processes including appetite regulation, metabolism and, more recently, dendritic spine architecture, long-term potentiation and cognition. Objective: The objective of this study was to determine the effects of two structurally non-peptide ghrelin receptor agonists (GSK894490A and CP-464709-18) on rodent cognition. Methods: All experiments were performed in male Lister hooded rats. Effects of the test compounds on rat cognitive performance was determined using the novel object recognition test, a modified water maze paradigm and a scopolamine-induced deficit in cued fear conditioning. These tests were chosen as they each probe a relatively independent cognitive domain and therefore potentially have differing underlying neural substrates. Results: Both compounds significantly improved performance in the novel object recognition and modified water maze tests but were unable to attenuate a scopolamine deficit in cued fear conditioning. Conclusions: These results demonstrate that the small-molecule ghrelin receptor agonists profiled here readily cross the blood/brain barrier and elicit pro-cognitive effects in recognition and spatial learning and memory tests. Based on these observations, the central ghrelin receptor would appear to be a chemically tractable receptor and perhaps should be considered as a new drug target for therapeutic approaches to treat diseases affecting cognition. .COPYRGT. 2009 Springer-Verlag.

L2 ANSWER 2 OF 21 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2009615220 EMBASE
TITLE: Hypoglycemia in the Emergency Department.
AUTHOR: Josefson, Jami, Dr. (correspondence)
CORPORATE SOURCE: Division of Pediatric Endocrinology, Northwestern University, Feinberg School of Medicine, Chicago, IL, United States. j-josefson@northwestern.edu
AUTHOR: Zimmerman, Donald
CORPORATE SOURCE: Division of Endocrinology, Children's Memorial Hospital, Chicago, IL, United States.
SOURCE: Clinical Pediatric Emergency Medicine, (December 2009) Vol. 10, No. 4, pp. 285-291.
Refs: 21
ISSN: 1522-8401 CODEN: CPEMBG
PUBLISHER: W.B. Saunders Ltd, 32 Jamestown Road, London, NW1 7BY, United Kingdom.
PUBLISHER IDENT.: S 1522-8401(09)00093-7
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
007 Pediatrics and Pediatric Surgery
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 11 Jan 2010
Last Updated on STN: 11 Jan 2010

AB Infants and children presenting to the emergency department with

hypoglycemia are a diagnostic emergency and require urgent treatment. The metabolic adaptive patterns of fasting occur earlier in children compared to adults, most notably with the development of ketone bodies. Glucose is the preferred energy source for the brain; however, when deprived of glucose, ketone bodies are an alternative fuel that may cross the blood-brain barrier. As infants and children have a relatively larger brain to body size and their rates of glucose use are higher, they are at increased risk of hypoglycemia. Collection of the "critical sample" to assist in the diagnostic work-up and urgent treatment to stabilize blood glucose levels is of paramount importance to protect the developing brain from glucose deprivation. .COPYRGT. 2009 Elsevier Inc. All rights reserved.

L2 ANSWER 3 OF 21 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2009694044 MEDLINE
DOCUMENT NUMBER: PubMed ID: 19765635
TITLE: The effect of peripheral administration of growth hormone on AD-like cognitive deficiency in NBM-lesioned rats.
AUTHOR: Doulah A H; Rohani A Haeri; Khaksari Haddad M; Motamedi F; Farbood Y; Badavi M; Malek M; Sarkaki A
CORPORATE SOURCE: Department of Biology, Sciences & Research Branch, Islamic Azad University (IAU), Poonak Squar, Ashrafi Isfehani High Way, Tehran, Iran.
SOURCE: Neuroscience letters, (2009 Nov 27) Vol. 466, No. 1, pp. 47-51. Electronic Publication: 2009-09-17. Journal code: 7600130. E-ISSN: 1872-7972. L-ISSN: 0304-3940.
PUB. COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200912
ENTRY DATE: Entered STN: 17 Oct 2009
Last Updated on STN: 29 Dec 2009
Entered Medline: 17 Dec 2009

AB This study aimed to evaluate the peripheral administration of growth hormone (GH) on AD-like cognitive deficiency in NBM-lesioned rats induced by ibotenic acid (5 microg/microl, in each side). Forty-eight male Wistar rats (20-24 months old; weighing 330+/-30 g) randomly divided into six groups (n=8). The groups include control group, which were intact rats; n-L+GH group: non-lesioned rats with GH treatment (1mg/kg, 9.00 am, for 10 consecutive days); n-L+Veh group: non-lesioned rats with vehicle treatment; L group: NBM-lesioned rats; L+GH group: NBM-lesioned rats with GH treatment and L+Veh group: NBM-lesioned rats with same volume of vehicle treatment. Peripheral administration of GH in control had no effect on learning and memory, while in L+GH group produced a significant enhancement in spatial learning and memory comparing to L and L+Veh groups. The percent of time spent in goal quarter during probe trial has decreased significantly in L and L+Veh groups compared to n-L groups. While it has increased significantly in L+GH group compared to L and L+Veh groups. No significant difference in percent of time spent was seen between the control and n-L groups. The GH has known as a mediate that effect through IGF-1. As the IGF-1 itself is earlier shown to improve cognitive function it is likely that the observed effect of GH is mediated through release of IGF-1 from peripheral tissue into the circulation for further transport across the BBB. This mechanism may result in the improvement of learning and memory in rats with NBM lesion.

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ACCESSION NUMBER: 2009008658 EMBASE
TITLE: Erythropoiesis-stimulating protein delivery in providing erythropoiesis and neuroprotection.
AUTHOR: Chang, Zhi-Yang
CORPORATE SOURCE: National Defense Medical Center, Graduate Institute of Life Sciences, Neihu, Taipei 114, Taiwan, Province of China.
AUTHOR: Chiang, Chiao-Hsi
CORPORATE SOURCE: National Defense Medical Center, School of Pharmacy, Neihu, Taipei 114, Taiwan, Province of China.
AUTHOR: Lu, Da-Wen
CORPORATE SOURCE: Tri-Service General Hospital, Department of Ophthalmology, No 325, Section 2, Chen-Kung Road, Neihu, Taipei 114, Taiwan, Province of China.
AUTHOR: Yeh, Ming-Kung, Prof. (correspondence)
CORPORATE SOURCE: Tri-Service General Hospital, National Defense Medical Center, Department of Clinical Pharmacy, No 325, Section 2, Chen-Kung Road, Neihu, Taipei 114, Taiwan, Province of China. mkyeh2004@gmail.com
SOURCE: Expert Opinion on Drug Delivery, (December 2008) Vol. 5, No. 12, pp. 1313-1321.
Refs: 73
ISSN: 1742-5247
PUBLISHER: Informa Healthcare, Telephone House, 69 - 77 Paul Street, EC2A 4LQ, United Kingdom.
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 008 Neurology and Neurosurgery
025 Hematology
029 Clinical and Experimental Biochemistry
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
039 Pharmacy
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jan 2009
Last Updated on STN: 16 Jan 2009

AB Erythropoietin (EPO), a glycoprotein, plays an important role in erythropoiesis and neuroprotection. EPO therapies for anemia or neurodegenerative diseases require frequent injections or high-dose systemic administration which may cause unwanted side effects. Various strategies for EPO delivery have been investigated for increasing EPO bioavailability and decreasing side effects, including nano/micro particles, PEGylation of EPO and transport-mediated delivery systems. Nano/micro particles provide EPO with long-term effect and protect EPO against proteolytic cleavage. PEGylated EPO prolong circulating time and reduce injection frequency of anemia treatment. A transport-mediated delivery system enables protein to cross biological barriers. Presently, there is no report about an effective delivery system of EPO for neuroprotection. This review focuses on EPO delivery systems for erythropoiesis or neuroprotection with prolonged duration and enhanced bioavailability. .COPYRGT. 2008 Informa UK Ltd.

L2 ANSWER 5 OF 21 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2008431818 EMBASE
TITLE: Ghrelin and ghrelin receptor inhibitors: Agents in the treatment of obesity.
AUTHOR: Soares, Joao-Bruno; Roncon-Albuquerque Jr., Roberto; Leite-Moreira, Adelino (correspondence)
CORPORATE SOURCE: University of Porto, Faculty of Medicine, Department of

Physiology, 4200-319 Porto, Portugal. amoreira@med.up.pt
SOURCE: Expert Opinion on Therapeutic Targets, (September 2008) Vol. 12, No. 9, pp. 1177-1189.
Refs: 90
ISSN: 1472-8222 CODEN: EOTTAO
PUBLISHER: Informa Healthcare, Telephone House, 69 - 77 Paul Street, EC2A 4LQ, United Kingdom.
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 029 Clinical and Experimental Biochemistry
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 19 Sep 2008
Last Updated on STN: 19 Sep 2008

AB Background: Current medical treatment of obesity is highly ineffective. Soon after its discovery as the endogenous ligand for the growth hormone secretagogue-receptor (GHS-R), ghrelin was shown to stimulate food intake (including in humans) and promote body weight gain and adipogenesis. Objectives: This review discusses the role of the ghrelin/GHS-R pathway in energy homeostasis regulation and its role as a novel molecular target for the treatment of obesity. Methods: Medline was searched for relevant articles published in English. Results/conclusion: A large series of animal studies shows that inhibition of the ghrelin/GHS-R pathway reduces food intake, body weight and adiposity, through reduction of appetite and augmentation of energy expenditure and fat catabolism. This suggests that inhibition of this novel pathway may be used to treat/prevent obesity and its complications. .COPYRGT. 2008 Informa UK Ltd.

ACCESSION NUMBER: 2007225628 EMBASE
TITLE: Experimental treatments for human transmissible spongiform encephalopathies: Is there a role for pentosan polysulfate?.
AUTHOR: Rainov, N.G., Dr. (correspondence)
CORPORATE SOURCE: Klinikum Augsburg, Department of Neurosurgery, Stenglinstr. 2, D-86156 Augsburg, Germany. nikolai.rainov@medizin.uni-halle.de
AUTHOR: Rainov, N.G., Dr. (correspondence)
CORPORATE SOURCE: Martin-Luther-University Halle-Wittenberg, Department of Neurosurgery, D-06122 Halle, Germany. nikolai.rainov@medizin.uni-halle.de
AUTHOR: Tsuboi, Y.
CORPORATE SOURCE: Fukuoka University, School of Medicine, Department of Neurology, Fukuoka, Japan.
AUTHOR: Krolak-Salmon, P.; Vighetto, A.
CORPORATE SOURCE: Hopital Neurologique, Department of Neurology, Lyon, France.
AUTHOR: Doh-ura, K.
CORPORATE SOURCE: Tohoku University, Graduate School of Medicine, Department of Prion Research, Sendai, Japan.
SOURCE: Expert Opinion on Biological Therapy, (May 2007) Vol. 7, No. 5, pp. 713-726.
Refs: 89
ISSN: 1471-2598 CODEN: EOBTA2
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 030 Clinical and Experimental Pharmacology
037 Drug Literature Index

038 Adverse Reactions Titles
039 Pharmacy
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 3 Jul 2007
Last Updated on STN: 3 Jul 2007

AB Human transmissible spongiform encephalopathies (TSEs), also known as prion diseases, are caused by the accumulation of an abnormal isoform of the prion protein in the CNS. Creutzfeldt-Jakob disease in its sporadic form is the most frequent type of human TSE. At present, there is no proven specific or effective treatment available for any form of TSE. Pentosan polysulfate (PPS) has been shown to prolong the incubation period when administered to the cerebral ventricles in a rodent TSE model. Cerebroventricular administration of PPS has been carried out in 26 patients with TSEs and has been shown to be well tolerated in doses ≤ 220 μg/kg/day. Proof of efficacy has been difficult because the specific and objective criteria for measurement of response have not been established yet. Preliminary clinical experience confirms extended survival in patients with variant Creutzfeldt-Jakob disease receiving intraventricular PPS; however, it is still not clear if this is due to PPS itself. Further prospective investigations of long-term intraventricular PPS administration are essential for the assessment of its effects. .COPYRGT. 2007 Informa UK Ltd.

L2 ANSWER 7 OF 21 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2007230701 EMBASE
TITLE: Nasal drug delivery system-factors affecting and applications.
AUTHOR: Jadhav, Kisan R. (correspondence); Gambhire, Manoj N.; Shaikh, Ishaque M.; Kadam, Vilasrao J.
CORPORATE SOURCE: Bharati Vidyapeeth's College of Pharmacy, Navi Mumbai 400 614, India. krj24@rediffmail.com
AUTHOR: Pisal, Sambjahi S.
CORPORATE SOURCE: Bharati Vidyapeeth Deemed University, Poona College of Pharmacy, Pune-411 038, India.
AUTHOR: Jadhav, Kisan R. (correspondence)
CORPORATE SOURCE: Department of Pharmaceutics, Bharati Vidyapeeth's College of Pharmacy, Navi-Mumbai-400614, India. krj24@rediffmail.com
SOURCE: Current Drug Therapy, (Jan 2007) Vol. 2, No. 1, pp. 27-38. Refs: 113
ISSN: 1574-8855
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 027 Biophysics, Bioengineering and Medical Instrumentation
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
039 Pharmacy
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 22 Jun 2007
Last Updated on STN: 22 Jun 2007

AB The nasal route is a potential alternative route for systemic availability of drugs restricted to intravenous administration, such as peptide and protein drugs and vaccines. This route is also advisable for drugs undergoing extensive first pass effect. Besides this, intranasal route has also been successfully exploited for bypassing the blood brain barrier [BBB] and subsequently
delivering drug molecules to central nervous system [CNS]. The present

article highlights the advantages, barriers, physicochemical factors and formulation related parameters affecting the nasal drug delivery. It also includes a note on nasal absorption enhancers. Also, the applications of nasal route for delivery of peptides and proteins, non-peptide drugs, vaccines and drug molecules to CNS have been summarized in depth. .COPYRGT. 2007 Bentham Science Publishers Ltd.

L2 ANSWER 8 OF 21 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2005558442 EMBASE
TITLE: Potential of growth hormone-releasing hormone antagonists (JV-1-36 and JV-1-42) for the treatment of brain tumours.
AUTHOR: Doggrell, Sheila A. (correspondence)
CORPORATE SOURCE: Division of Health Practice, Auckland University of Technology, Akoranga Campus, Northcote, Auckland, New Zealand. s.doggrell@xtra.co.nz
SOURCE: Expert Opinion on Investigational Drugs, (Dec 2005) Vol. 14, No. 12, pp. 1561-1564.
Refs: 13
ISSN: 1354-3784 CODEN: EOIDER
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 12 Jan 2006
Last Updated on STN: 12 Jan 2006

AB Malignant glioblastoma is the most common type of primary brain tumour in adults. Current therapies are palliative and prolong quality survival. JV-1-36 and JV-1-42 are examples of peptide growth hormone-releasing hormone antagonists. JV-1-36 has been shown to decrease the size of subcutaneous human glioblastoma tumours in nude mice, and to extend the survival of nude mice implanted orthotopically with glioblastoma. JV-1-42 has been shown to cross the blood-brain barrier very effectively in mice; thus, the JV series of growth hormone-releasing hormone antagonists are showing promise for glioblastoma and should be further developed for the treatment of this condition. .COPYRGT. 2005 Ashley Publications.

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ACCESSION NUMBER: 2004250472 EMBASE
TITLE: Antiaging methods and medicines for the memory.
AUTHOR: Banks, William A., Dr. (correspondence); Farr, Susan A.
CORPORATE SOURCE: Division of Geriatric Medicine, Department of Internal Medicine, S. Louis Univ. School of Medicine, 1402 South Grand Boulevard, St. Louis, MO 63104, United States. bankswa@slu.edu
AUTHOR: Banks, William A., Dr. (correspondence); Farr, Susan A.
CORPORATE SOURCE: Geriatric Res., Educ., Clin. Ctr., St. Louis Vet. Aff. Medical Center, 915 North Grand Avenue, St. Louis, MO 63106, United States. bankswa@slu.edu
SOURCE: Clinics in Geriatric Medicine, (May 2004) Vol. 20, No. 2, pp. 317-328.
Refs: 79
ISSN: 0749-0690 CODEN: CGMEE6
PUBLISHER IDENT.: S 0749-0690(04)00024-2

COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 020 Gerontology and Geriatrics
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 1 Jul 2004
Last Updated on STN: 1 Jul 2004

AB The interaction of bedside and basic science has led to the identification of a short list of pathological proteins as causal in Alzheimer's disease. AssP has received the most attention, and work with animal models has reinforced the evidence that overproduction of AssP causes cognitive impairments. Animal models are now being used to discover and develop unique therapeutics directed at reversing the deleterious effects of AssP. These models strongly suggest that established Alzheimer's disease might be reversible, not just preventable. Animal models are also demonstrating that other peptides and proteins can enhance or impair cognitive function. These peptides and proteins add further to the list of possible therapeutic candidates. Approaches such as these, and not the commercial antiaging remedies that have no scientific basis [75-79], will eventually provide medicine for memory enhancement.

L2 ANSWER 10 OF 21 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 1999243380 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10226793
TITLE: Insulin-like growth factor-1 and growth hormone (GH) levels in canine cerebrospinal fluid are unaffected by GH or GH secretagogue (MK-0677) administration.
AUTHOR: Prahalada S; Block G; Handt L; DeBurlet G; Cahill M; Hoe C M; van Zwieten M J
CORPORATE SOURCE: Department of Safety Assessment, Merck Research Laboratories (MRL), West Point.. srinivasa_prahalada@merck.com
SOURCE: Hormone and metabolic research = Hormon- und Stoffwechselforschung = Hormones et metabolisme, (1999 Feb-Mar) Vol. 31, No. 2-3, pp. 133-7. Journal code: 0177722. ISSN: 0018-5043. L-ISSN: 0018-5043.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 12 Jul 1999
Last Updated on STN: 12 Jul 1999
Entered Medline: 22 Jun 1999

AB Elevation in circulating GH levels results in a dose-related increase in serum insulin-like growth factor-1 (IGF-1) levels in dogs. However, it is not known whether elevations in systemic IGF-1 and GH levels contribute to the cerebrospinal fluid (CSF) levels of these hormones. Therefore, a study was designed in dogs to determine if elevated circulating GH levels was a result of a GH secretagogue (MK-0677) or if exogenous GH administration resulted in increased IGF-1 and GH levels in the CSF of dogs. A total of 12 normal, young adult male dogs were randomized to three treatment groups (4 dogs/group) based on body weight. There were 4 vehicle control dogs. A group of 4 dogs were dosed orally with MK-0677 (5 mg/kg/day) dissolved in deionized water. A third group of 4 dogs received subcutaneous injections of porcine GH (pGH) at a dose of

0.1 IU/kg/day. From all dogs, blood and CSF samples were collected prior to the initiation of treatment and on days 7 and 15 of treatment. All samples were assayed using a validated radioimmunoassay. Administration of MK-0677 or pGH resulted in a statistically significant (P < or = 0.05) increased body weight gain and increased serum IGF-1 and GH levels. In contrast, administration of MK-0677 resulted in no significant (P > 0.05) increase in CSF IGF-1 or GH levels on days 7 or 15 of the study. The CSF IGF-1 values ranged from 1.2 to 2.0 ng/ml with minimal variation among three separate samples taken during the course of the study from each dog. Similarly, the CSF GH levels were very low (< 0.98 ng/ml to 2.4 ng/ml) in all dogs irrespective of treatment group. This study has demonstrated that there is no correlation between the circulating levels of IGF-1 or GH and the levels of these hormones in the CSF of normal dogs. An approximately 100-fold difference between serum and CSF IGF-1 levels in vehicle control dogs suggest that there is a blood-brain barrier for the circulating IGF-1. Similarly, failure to see an elevation in CSF GH levels despite increases in serum GH levels shows that there is a blood-brain barrier for GH in normal dogs. These results suggest that the likely source of GH and IGF-1 in the CSF of dogs is from the CNS.

L2 ANSWER 11 OF 21 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1999181169 EMBASE
TITLE: Blood-brain barrier for human growth hormone and insulin-like growth factor-I.
AUTHOR: Coculescu, Mihail, Dr. (correspondence)
CORPORATE SOURCE: Inst. of Endocrinology C.I. Parhon, Bucharest, Romania.
AUTHOR: Coculescu, Mihail, Dr. (correspondence)
CORPORATE SOURCE: Department of Endocrinology, UMF C. Davila, PO Box 41-31, 78260 Bucharest, Romania.
AUTHOR: Coculescu, Mihail, Dr. (correspondence)
CORPORATE SOURCE: Department of Endocrinology, PO Box 41-31, 78260 Bucharest, Romania.
SOURCE: Journal of Pediatric Endocrinology and Metabolism, (1999) Vol. 12, No. 2, pp. 113-124.
Refs: 82
ISSN: 0334-018X CODEN: JPEMFT
COUNTRY: Israel
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 003 Endocrinology
037 Drug Literature Index
007 Pediatrics and Pediatric Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 10 Jun 1999
Last Updated on STN: 10 Jun 1999

AB There is a blood-brain barrier (BBB) for GH. A certain, unknown amount of GH passes the BBB, acts on the neuronal GH receptors and directly influences the brain mechanisms serving the feedback and ultradian secretion of GH. The high density of GH receptors in the choroid plexus suggests a possible receptor-mediated transcytosis transport. The effects of GH on brain development, neuronal plasticity and neuroprotection seem to be mediated by IGFs. GH and IGFs are also synthesized in the brain. The relative contributions to brain functions of GHs produced inside and outside the BBB are unknown. The cerebrospinal fluid (CSF) space is the compartment inside the barrier accessible to clinicians. High GH levels in CSF were reported in acromegaly and also a small increase was

reported after chronic administration of hGH in GH-deficiency syndromes. For the practitioner it is necessary to determine the normal range of hGH levels in CSF.

L2 ANSWER 12 OF 21 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 1999046348 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9828908
TITLE: L-5-hydroxytryptophan does not stimulate LH secretion directly from the pituitary in patients with gonadotrophin releasing hormone deficiency.
AUTHOR: Lado-Abeal J; Grana M; Rey C; Cabezas-Cerrato J
CORPORATE SOURCE: Endocrinology and Nutrition Service, Galician General Hospital, Spain.. cbbjjl@wpoffice.net.ttuhsc.edu
SOURCE: Clinical endocrinology, (1998 Aug) Vol. 49, No. 2, pp. 203-7.
Journal code: 0346653. ISSN: 0300-0664. L-ISSN: 0300-0664.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199812
ENTRY DATE: Entered STN: 15 Jan 1999
Last Updated on STN: 15 Jan 1999
Entered Medline: 3 Dec 1998

AB OBJECTIVE: There is abundant histological and physiological evidence that serotonin plays a role in the regulation of LH secretion in rats. Studies in human subjects have been few, but their results include the finding that pulsatile administration of L-5-hydroxytryptophan (5-HTP, the immediate precursor of serotonin) amplifies LH secretion in women in the medium-late follicular phase, and that this effect is not due to 5-HTP directly inducing LH secretion by the pituitary. We have investigated whether 5-HTP amplifies LH secretion by enhancing the response of the pituitary to GnRH. PATIENTS: Seven patients aged 20-40 years with hypogonadotrophic hypogonadism (HH) of hypothalamic origin (3 men with Kallmann's syndrome, 2 women without anosmia and with GH deficiency, and 2 women with anorexia nervosa). DESIGN: To prime the pituitary, subcutaneous pulsatile GnRH was administered for 7 days at the rate of one 5-20 micrograms pulse every 90 min. The day before the investigation, this regimen was replaced by 1.5-3 micrograms intravenous pulses at the same frequency. On the day of the investigation, 3 ml blood samples were taken every 10 min from 0850 to 19:00 hours. After the first two samples, the intravenous GnRH pulse frequency was increased to one per hour and was maintained at this level throughout the rest of the study. The first 4 h of the study acted as a control phase allowing determination of the pituitary response to GnRH. At 1300 h, 75 mg of the aromatic-L-amino-acid decarboxylase inhibitor carbidopa was administered orally; carbidopa does not cross the blood-brain barrier, and prevents peripheral conversion of 5-HTP to serotonin. At 1600 h, another 75 mg dose of carbidopa was administered, and administration of 8-20 mg pulses of 5-HTP at a rate of one pulse per hour was begun. MEASUREMENTS: LH was determined in triplicate by an immunoradiometric assay (IRMA), and LH pulses identified by means of a program developed in our laboratory. RESULTS: When pulsatile administration of GnRH was accompanied by administration of carbidopa and 5-HTP, LH pulse amplitude (2.32 +/- 0.71 IU/l) did not differ significantly from its value in either the GnRH+ carbidopa phase (2.58 +/- 1.12 IU/l) or the unaccompanied GnRH phase (2.77 +/- 1.76 IU/l). CONCLUSIONS: L-5-hydroxytryptophan-induced amplification of LH secretion in humans is not due to enhancement of the pituitary response to GnRH. The effect of L-5-hydroxytryptophan must therefore be

due to its action on the hypothalamus, where it may be hypothesized that it increases GnRH release.

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ACCESSION NUMBER: 1996352194 EMBASE
TITLE: Expansion and shrinkage of central nervous system tumor coinciding with human growth hormone therapy: Case report.
AUTHOR: Connors, Matthew H., Dr. (correspondence); Kollipara, Sobha
CORPORATE SOURCE: Departments of Pediatrics, University of California, Davis Medical Center, Sacramento, CA, United States.
AUTHOR: Boggan, James E.
CORPORATE SOURCE: Department of Neurosurgery, University of California, Davis Medical Center, Sacramento, CA, United States.
AUTHOR: Chong, Brian
CORPORATE SOURCE: Department of Radiology, University of California, Davis Medical Center, Sacramento, CA, United States.
AUTHOR: Connors, Matthew H., Dr. (correspondence)
CORPORATE SOURCE: Department of Pediatrics, University of California, Davis, 2516 Stockton Blvd., Sacramento, CA 95817, United States.
AUTHOR: Connors, Matthew H., Dr. (correspondence)
CORPORATE SOURCE: Department of Pediatrics, University of California, 2516 Stockton Blvd., Sacramento, CA 95817, United States.
SOURCE: Neurosurgery, (Dec 1996) Vol. 39, No. 6, pp. 1243-1246. Refs: 10
ISSN: 0148-396X CODEN: NRSRDY
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 037 Drug Literature Index
007 Pediatrics and Pediatric Surgery
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 18 Dec 1996
Last Updated on STN: 18 Dec 1996

AB OBJECTIVE AND IMPORTANCE: The influence of human growth hormone (hGH) therapy on the recurrence rates of childhood central nervous system tumors is controversial. Because growth hormone has the ability to increase cell proliferation, it is recommended that hGH therapy wait until central nervous system lesions are inactive and antitumor therapy complete, usually 1 to 2 years. CLINICAL PRESENTATION: We report the enlargement and decrease in size of a hypothalamic pilocytic astrocytoma in a 12-year-old boy after two trials of hGH. Partial resection and radiation of the tumor were performed at 3 years of age, with no change noted over the next 9 years. His height was less than the 5th centile with midparental height at the 90th to 95th centiles. Growth velocity was 3.3 cm/yr. Bone age was normal and there were no signs of puberty. There was no GH response to clonidine and L-dopa testing. INTERVENTION: Volume measurements were performed on gadolinium enhanced tumor images. Growth rate increased to 11.7 cm and the tumor volume increased 230% over the 12 months of hGH therapy. Significant tumor shrinkage (42%) and growth deceleration occurred within the 3 month interval of stopping hGH. Tumor volume again increased (134%) and decreased (22%) after restarting and then stopping hGH. No evidence of tumor necrosis or alteration in ventricular size was found. The patient was asymptomatic. CONCLUSION: These observations indicate that tumor size change is associated with the metabolic response to hGH therapy. It is unclear whether the volume increase represents altered blood-brain or selective blood-tumor barrier permeability, growth factor receptors, and/or tumor cell growth.

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ACCESSION NUMBER: 1994007040 EMBASE
TITLE: Growth hormone secretion in response to surgery. Effects of cholinergic blockade and octreotide.
AUTHOR: Desborough, J.P.; Griffin, R.A.; Moore, C.M.; Burrin, J.M.; Hall, G.M.
CORPORATE SOURCE: St. George's Hospital Medical School, Department of Anaesthesia, Cranmer Terrace, Tooting, London SW17 0RY, United Kingdom.
AUTHOR: Desborough, J., Dr. (correspondence)
CORPORATE SOURCE: St. George's Hospital Medical School, Department of Anaesthesia, Cranmer Terrace, Tooting, London SW17 0RY, United Kingdom.
SOURCE: Hormone and Metabolic Research, (1993) Vol. 25, No. 12, pp. 640-643.
ISSN: 0018-5043 CODEN: HMMRA2
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 30 Jan 1994
Last Updated on STN: 30 Jan 1994

AB Cholinergic blockade markedly reduces the growth hormone (GH) response to most stimuli, with the exception of insulin induced hypoglycaemia. We have administered the cholinergic antagonist, atropine, known to cross the blood brain barrier, to eight healthy female patients prior to elective surgery, in order to investigate the role of cholinergic pathways in the GH response to surgery. Additionally, eight patients received the octapeptide analogue of somatostatin, octreotide, known to suppress GH secretion. A control group matched for age and weight received no injection. The GH response to surgery was assessed by peak values and areas under curves. Octreotide resulted in a significant inhibition of GH secretion compared with the control group ($p < 0.01$ for both parameters). In contrast, atropine did not significantly inhibit the GH response to surgery. In conclusion, octreotide completely suppressed GH secretion during surgery, whereas cholinergic blockade was ineffective. Thus surgery is similar to insulin induced hypoglycaemia in that the GH response is not decreased by cholinergic blockade.

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ACCESSION NUMBER: 1992111933 EMBASE
TITLE: Antidepressant challenge tests: The interface of pharmacokinetics and pharmacodynamics.
AUTHOR: Golden, R.N.; Gilmore, J.H.; Carson, S.W.
CORPORATE SOURCE: Psychiatry Department, Medical School Wing B, University of North Carolina, Campus Box 7160, 215 South Wing Drive, Chapel Hill, NC 27599-7160, United States.
SOURCE: Psychopharmacology Bulletin, (1991) Vol. 27, No. 4, pp. 611-617.
ISSN: 0048-5764 CODEN: PSYBB9
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 003 Endocrinology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English

SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 8 May 1992
Last Updated on STN: 8 May 1992

AB Antidepressant compounds have been used as probes in neuroendocrine challenge tests designed to assess the functional responsivity of central neurotransmitter systems. Neurohormonal responses to the antidepressants desipramine and clomipramine, for example, can provide information regarding noradrenergic and serotonergic function, respectively. Pharmacokinetic factors can affect these antidepressant challenge tests. Proper attention to pharmacokinetic issues in the design, implementation, and analysis of data obtained with these probes will provide a more clear understanding of the pharmacodynamic meaning of the results from studies that apply antidepressant challenges.

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ACCESSION NUMBER: 1991285932 EMBASE
TITLE: Evaluation of the sites of opioid influence on anterior pituitary hormone secretion using a quaternary opiate antagonist.
AUTHOR: Simpkins, J.W. (correspondence); Swager, D.; Millard, W.J.
CORPORATE SOURCE: Department of Pharmacodynamics, College of Pharmacy, University of Florida, Gainesville, FL, United States.
AUTHOR: Simpkins, J.W. (correspondence)
CORPORATE SOURCE: J. Hillis Miller, Health Center, University of Florida, Gainesville, FL 32610, United States.
SOURCE: Neuroendocrinology, (1991) Vol. 54, No. 4, pp. 384-390. ISSN: 0028-3835 CODEN: NUNDAJ
COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 16 Dec 1991
Last Updated on STN: 16 Dec 1991

AB Studies were conducted to determine the effects of a potent narcotic antagonist, nalmefene methyliodide, which does not cross the blood -brain barrier (BBB), on the secretion of anterior pituitary hormones and on the anterior pituitary hormonal response to morphine sulfate. Since the localization of opiate receptor responses to inside or outside the BBB depended upon the relative ability of nalmefene HCl and nalmefene methyliodide to penetrate the BBB, initial studies were conducted to document that nalmefene methyliodide does not block opiate receptors inside the central nervous system. While nalmefene HCl blocked morphine-induced antinociceptive responses at doses as low as 10 μg/kg, nalmefene methyliodide was ineffective in this regard at doses as high as 500 μg/kg. The luteinizing hormone (LH) suppression and prolactin (PRL) secretion induced by morphine was blocked by both nalmefene HCl and its methyliodide analogue, indicating that the opioid receptor type which mediates both responses is located outside the BBB. We observed that basal PRL levels were reduced by nalmefene HCl but not by nalmefene methyliodide indicating that basal PRL secretion is influenced by opioid neurons inside the BBB. While nalmefene HCl blocked morphine-induced suppression of thyroid-stimulating hormone (TSH) release, nalmefene methyliodide was less effective, suggesting that opiate-induced TSH suppression may be mediated by receptors located both within and outside the BBB. Nalmefene HCl caused a growth hormone (GH)-secretory response by itself, but nalmefene HCl and nalmefene methyliodide were ineffective in blocking morphine-induced GH secretion. These data indicate that opiate

effects on LH and PRL secretion are mediated by receptors located outside the BBB, while the location of opiate receptors which influence TSH and GH secretion could not be defined.

L2 ANSWER 17 OF 21 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 1987232475 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3035598
TITLE: The effects of the systemic administration of N-methylmorphine chloride, a quaternary analogue of morphine that does not cross the blood-brain barrier, on the release of anterior pituitary hormones in the rat.
AUTHOR: Pechnick R N; George R; Poland R E
SOURCE: Psychoneuroendocrinology, (1987) Vol. 12, No. 1, pp. 67-71. Journal code: 7612148. ISSN: 0306-4530. L-ISSN: 0306-4530.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198706
ENTRY DATE: Entered STN: 5 Mar 1990
Last Updated on STN: 5 Mar 1990
Entered Medline: 26 Jun 1987
AB The acute administration of morphine elicits changes in the release of anterior pituitary hormones. The locus of this action is thought to be in the central nervous system, specifically the hypothalamus. There are some data suggesting that systemically administered opiates and opioid peptides can act outside of the blood-brain barrier to influence anterior pituitary hormone release. To test this hypothesis we examined the effects of the systemic administration of N-methylmorphine chloride, a quaternary analogue of morphine that does not cross the blood-brain barrier, on the release of corticosterone, growth hormone, prolactin, luteinizing hormone, and thyroid stimulating hormone in the rat. N-methylmorphine caused increases in the release of growth hormone and prolactin, but serum levels of corticosterone, luteinizing hormone and thyroid stimulating hormone were unaffected. These results indicate that the opiate-induced release of growth hormone and prolactin may be mediated in part by sites outside of the blood-brain barrier.

L2 ANSWER 18 OF 21 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1986251850 EMBASE
TITLE: Inhibitory effect of antimuscarinic cholinergic drug (atropine) on growth hormone (GH) secretion induced by GH-releasing factor.
AUTHOR: Yagi, H.; Nagashima, K.; Noji, T.; et. al.
CORPORATE SOURCE: Department of Pediatrics, Gunma University School of Medicine, Maebashi, Gunma 371, Japan.
SOURCE: Hormone and Metabolic Research, (1986) Vol. 18, No. 10, pp. 723-724.
ISSN: 0018-5043 CODEN: HMMRA2
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
008 Neurology and Neurosurgery
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1991

Last Updated on STN: 10 Dec 1991

AB We investigated whether atropine, a muscarinic cholinergic antagonist, affects the GRF-stimulated GH secretion and the concentration of peripheral plasma SRIF. Our study showed that the GH release induced by GRF administration was completely inhibited, and that the level of SRIF in peripheral blood was elevated by atropine in six normal men. As atropine can cross the blood brain barrier , there are two possibilities; the depression of GH secretion by atropine is the direct action on somatotrophs, or the indirect effect of enhanced SRIF secretion in hypothalamus. However, the elevation of SRIF level was delayed 30 or 60 min when GH suppression by atropine had already occurred. Therefore, it was difficult to explain the depression of GH secretion by the antagonizing effect of SRIF. It is also unclear whether the measured SRIF in peripheral blood originated from the hypothalamus or extrahypothalamic areas such as pancreas or gut. Although a further evaluation on the SRIF-releasing activity of atropine itself and the origin of SRIF will be required, it is possible to conclude at present that the suppressed hGRF-induced GH secretion and the elevation of SRIF in peripheral blood after the administration of atropine, are independent phenomena and that atropine directly inhibits the GH release from pituitary gland by hGRF as indicated by Massara et al.

L2 ANSWER 19 OF 21 MEDLINE on STN DUPLICATE 5

ACCESSION NUMBER:	1986069016 MEDLINE
DOCUMENT NUMBER:	PubMed ID: 2999871
TITLE:	Corticotropin releasing factor: basic studies and clinical applications.
AUTHOR:	Chrousos G P; Calabrese J R; Avgerinos P; Kling M A; Rubinow D; Oldfield E H; Schuermeyer T; Kellner C H; Cutler G B Jr; Loriaux D L; et al
SOURCE:	Progress in neuro-psychopharmacology & biological psychiatry, (1985) Vol. 9, No. 4, pp. 349-59. Ref: 22 Journal code: 8211617. ISSN: 0278-5846. L-ISSN: 0278-5846.
PUB. COUNTRY:	ENGLAND: United Kingdom
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE) General Review; (REVIEW)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals
ENTRY MONTH:	198601
ENTRY DATE:	Entered STN: 21 Mar 1990 Last Updated on STN: 29 Jan 1999 Entered Medline: 22 Jan 1986

AB Corticotropin releasing factor (CRF) is a newly sequenced peptide first isolated from sheep hypothalami and thought to be an important modulator of both the pituitary-adrenal axis and the sympathetic nervous system. We administered intravenous, intramuscular, and intracerebroventricular CRH to non-human primates and measured plasma ACTH, beta endorphin, cortisol, GH and PRL responses to CRF. In addition, we determined the pharmacokinetic properties of I125 in these primates. We administered CRF as an intravenous bolus or as a continuous infusion to normal volunteers and as an intravenous bolus to patients with disorders of the hypothalamic-pituitary-adrenal axis, such as Cushing's syndrome and adrenal insufficiency, and patients with endogenous depression and mild hypercortisolism, and assessed their plasma ACTH, cortisol, GH and PRL responses. In addition, we determined the pharmacokinetic properties of CRF in man by measuring CRF immunoreactivity in plasma. CRF given intravenously to primates or man is a slowly metabolized, long-acting, secretagogue of ACTH, beta-endorphin and cortisol. When given intracerebroventricularly to primates it stimulates the hypothalamic-pituitary-adrenal axis without escaping into the plasma and it is actively cleared in the CNS. It does not cross the

blood brain barrier appreciably when given intravenously. CRF given to primates and men as an intravenous continuous infusion has only mild ACTH stimulating effects and this may be due to an intact cortisol negative feedback system. Finally, CRF causes characteristic plasma hormone responses in patients with Cushing's disease, adrenal insufficiency and depression.

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ACCESSION NUMBER: 1982182616 EMBASE
TITLE: Pharmacokinetics of α-difluoromethyl ornithine in rat brain and in intracranial and subcutaneous gh tumors.
AUTHOR: Levin, V.A.; Csejtey, J.; Blank, D.
CORPORATE SOURCE: Brain Tumor Res. Cent., Univ. California, San Francisco, CA 94143, United States.
SOURCE: Proceedings of the American Association for Cancer Research, (1982) Vol. Vol. 23, pp. No. 814.
CODEN: PAACA3
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991

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ACCESSION NUMBER: 1982193255 EMBASE
TITLE: Effect of domperidone on apomorphine-induced growth hormone secretion in normal men.
AUTHOR: Lal, S.; Nair, N.P.V.; Iskander, H.L.; et. al.
CORPORATE SOURCE: Douglas Hosp. Res. Cent., Douglas Hosp., Verdun, Que. H4H 1R3, Canada.
SOURCE: Journal of Neural Transmission - General Section, (1982) Vol. 54, No. 1-2, pp. 75-84.
CODEN: JNTMAH
COUNTRY: Austria
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
003 Endocrinology
037 Drug Literature Index
008 Neurology and Neurosurgery
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991

AB Domperidone, a peripheral dopamine (DA) receptor blocker which poorly crosses the blood-brain barrier and which is inactive towards dopamine-sensitive adenylate cyclase, in a dose (100 μg/kg) sufficient to increase serum prolactin levels at least 5-fold, decreased the growth hormone (GH) response to the DA receptor agonist, apomorphine HCl (Apo) (0.5 mg s.c.) in each of six normal men examined. The mean GH increment at 30, 45, 60 and 75 min following Apo injection, the mean individual peak increment and the mean individual GH secretion (ng min) was significantly decreased by domperidone pretreatment ($p<0.05$-$p<0.02$). These results indicate that in man Apo stimulates GH secretion by an effect on DA receptors which are not linked to adenylate cyclase and which are situated at a locus in the hypothalamic-pituitary axis that lies outside the blood-brain barrier.

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                AND (INTRAMUSC? OR SUBCUTANEOUS?) AND (BLOOD(W) BRAIN(W)
                BARRIER OR BBB)
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